



**ORIGINAL RESEARCH PAPER**

**Physiology**

**STUDY OF EFFECT ON AUTONOMIC NERVOUS SYSTEM IN SMOKERS AND NON SMOKERS**

**KEY WORDS:** Autonomic nervous system, smoking, respiratory rate, chronic obstructive pulmonary diseases.

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**ABSTRACT**

Smoking(Tobacco) is a major cause of death worldwide. Cigarette smoke components are mainly carbon monoxide, carbon dioxide, nitrogen oxides, ammonia, volatile nitrosamines, hydrogen cyanide, volatile sulphur containing compounds, volatile hydrocarbons, alcohols, aldehydes and ketones. Some of these compounds affect ciliary movement in the lungs and autonomic nervous system. Habitual cigarette smoking affects respiratory system and cardio vascular system through autonomic nervous system. So we conducted a study to compare the smoking effect on autonomic nervous system in smokers and non-smokers. We found that Pulse Pressure, Systolic and Diastolic BP (Hg) was significantly higher in smokers as compared to non smokers. Respiratory Rate (RR) and Peak Expiratory Flow Rate (PEFR) were lower in smokers as compared to non smokers, the difference being statistically significant. Significant increase in diastolic blood pressure in habitual smokers with isometric hand grip exercise test was found indicating movement of sympathetic nervous system. The RR and PEFR were lower in smokers than the non smokers indicating Chronic Obstructive Pulmonary Disease.

**Introduction**

Long term smoking induces down regulation of the beta adrenergic receptors. Plasma catecholamine levels are significantly higher in smokers than non-smokers as explained by beta blockers which are less effective in the treatment of hypertension in smokers as compared to nonsmokers. Such observations indicate that cigarette smoking might induce a beta adrenergic tone with considerable impact on cardiovascular regulations. Further these results imply that even if beta receptor function per second is unaltered in cigarette smokers their down regulation of beta receptor results in relative increase in alpha adrenergic tone at any given catecholamine concentration. A decrease in PEFR in smokers significantly increases progressive deterioration of the lung functions and is a risk factor in chronic obstructive pulmonary disease, in which pulmonary mechanism deteriorates and causes loss of elastic recoil of the lungs and slows down the forced expiration with nicotine, and causes the mucus-producing cells to grow in size and number.

**Material and Methods**

Our study was carried out in the Department of Physiology at RIMS, RAIPUR, CG, from 2014 to 2015 after approval of institutional ethical committee. About 83 male individuals were randomly selected from the Medicine outdoor department of the hospital out of which 59 were habitual cigarette smokers who consumed at least 10 cigarettes per day and 24 were non smokers of age ranging from 20 to 50 years. A structured performa was designed to evaluate and record the personal data of the

selected subjects regarding their name, age, sex, height and weight, personal history like smoking, with duration and quantity, any history of lung disease, history of persistent cough etc. Individuals with history of hypertension, allergy and diabetics, autonomic dysfunctions were excluded. For each subject (both smoker and non smoker) the respiratory rate (cycles/min), heart rate (beats/min) and blood pressure using sphygmomanometer was recorded. The parasympathetic activity was assessed by heart rate response to Valsalva manoeuvre. Each subject was told to perform Valsalva manoeuvre for 15 seconds by blowing into a mouth piece attached to a sphygmomanometer and maintain a pressure of 40 mm Hg for 15sec. Three trials were performed at intervals of 5 minutes. A continuous ECG was recorded 1 minute before the maneuver (resting period), during maneuver (strain period, 15 seconds) and 60 seconds subsequently after the strain period. Valsalva ratio was taken as the maximum ratio of

maximum R-R interval after the strain to that of shortest R-R interval during the strain. The sympathetic activity was assessed by blood pressure response to sustained hand grip. The subject was asked to sit comfortably in chair. Initially the subject was asked to exert maximal strength on hand grip dynamometer with right hand. First the maximum voluntary contraction (MVC) was determined and then the subject was asked to exert 30% of MVC for 5 minutes with right hand. Diastolic blood pressure was measured in left hand at rest and at 1 minute interval during handgrip. The maximum rise of diastolic pressure during 30% of MVC over the resting diastolic blood pressure was noted. Peak Expiratory Flow Rate (PEFR): It is the maximum velocity (liters/minute) with which air is forced out of the lungs in a single forced expiratory effort. The subject should be in sitting position; nose closed with the help of clip and should take a deep breath and then place the mouth piece. The subject should then blow out as hard as possible in :-a short sharp blast. Three readings were taken after an adequate period of rest between each attempt. The maximal values were taken. The data was statistically analyzed using the SPSS software (version 12.0) and by applying Student's t-test.

**RESULT**

There was no significant difference in their age, weight and BMI. Also, the Pulse, DBP, MAP, RR (Shortest), RR interval Shortest (sec) and RR ratio in smokers was high as compared to non smokers, the difference being statistically insignificant. RR longest, RR interval (sec) in smokers was lower as compared to non smokers and was not significant. SBP in smokers was significantly higher as compared to non smokers.

**Table-1: Anthropometric parameters of smokers and non-smokers**

ANTHROPOMETRIC SMOKERS	SMOKERS (n=59) MEAN +- SD	NON SMOKERS (n=24) MEAN+-SD	P VALUE
AGE (Yr.)	36.47+8.12	35.33+7.18	0.55
WEIGHT(kg)	68.51+9.99	66.75+10.19	0.47
HIGHT(m)	1.69+0.08	1.69+0.10	0.86
BMI(kg/m2)	24.04+3.84	23.53+4.05	0.59

**DISCUSSION**

**Our study** was carried out in 59 habitual smokers and 24 non smokers who served as controls for testing autonomic functions. Evaluation of status of autonomic nervous system was done with the help of two non invasive tests like Valsalva maneuver and

sustained hand grip. Our study is almost related to the study of Ewing et al who observed that during sustained hand grip, there was a sharp rise in diastolic pressure due to increase in peripheral vascular resistance. Our results revealed a significant rise in blood pressure in smokers as compared to non smokers. This is in accordance with the observations reported by previous studies. Benowitz et al conducted a study among smokers and nicotine gum users and concluded that prominent cardiovascular effects of nicotine were result of activation of sympathetic nervous system increasing heart rate and blood pressure. Grassi et al reported the mechanism responsible for sympathetic activation due to cigarette smoking was associated with plasma catecholamines suggesting an adrenergic stimulation. In our study also there is an increase in blood pressure in smokers as compared to non smokers which could be explained due to increased sensitivity of sympathetic nervous system due to the stimulating tests (Sustained Hand grip). Gerhardt et al studied the effects of smoking on baroreceptors in smokers and reported an increased blood pressure among them as compared to non smokers. These findings are similar to our results. The present study revealed a significant decrease in PEFr in smokers as compared to non smokers. Suzuki et al also concluded that smokers had lower PEFr as compared to non smokers.

### Conclusion

Habitual cigarette smoking affects respiratory system and cardiovascular system through autonomic nervous system. Our observations revealed that there was a significant increase in diastolic blood pressure in habitual smokers with isometric hand grip exercise test indicating movement of sympathetic nervous system. Respiratory rate and PEFr was lower in smokers as compared to the non smokers indicating Chronic Obstructive Pulmonary Disease. The RR and PEFr were lower in smokers than the non smokers indicating Chronic Obstructive Pulmonary Disease.

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